it not be appropriate to use beta-blocker therapy in addition to implantable devices to reduce the risk of ventricular tachyfibrillation in light of the published data demonstrating reduced mortality as well as sudden death in those receiving beta-blocker intervention?

Charles L. Baird, Jr., MD
Virginia Heart Institute
Richmond, Virginia

PII S0735-1097(99)00292-2

REFERENCE

1. Pires LA, Lehmann MH, Steinman RT, et al. Sudden death in implantable cardioverter-defibrillator recipients: clinical context, arrhythmic events and device responses. J Am Coll Cardiol 1999;33:24–32.

REPLY

We read Dr. Baird's comments with much interest. He raised two important issues regarding our findings (1). First, with respect to the effect of possible proarrhythmia from antiarrhythmic drugs, it is certainly plausible that some of the deaths may have been related to drug proarrhythmia. Indeed, one death (patient no. 2, Table 1) was attributed to potential adverse drug (tocainide and encainide) effect; however, whether or not other deaths were related to drug proarrhythmia could not be ascertained from our data. Second, the use of beta-blocker therapy certainly should be encouraged in the proper settings. The benefit of beta-blocker therapy is undisputed in patients at risk for arrhythmic events (e.g., after myocardial infarction) and may, in fact, be beneficial as single or adjuvant therapy in select patients who have had symptomatic, sustained ventricular arrhythmias (2,3).

Luis A. Pires, MD
Cardiac Electrophysiology
St. John Hospital and Medical Center
Detroit, Michigan

PII S0735-1097(99)00293-4

REFERENCES

1. Pires LA, Lehmann MH, Steinman RT, et al. Sudden death in implantable cardioverter-defibrillator recipients: clinical context, arrhythmic events and device responses. J Am Coll Cardiol 1999;33:24–32.
2. Steinbeck G, Andresen D, Bach P, et al. A comparison of electrophysiologically guided antiarrhythmic drug therapy with beta-blocker in patients with symptomatic, sustained ventricular tachyarrhythmias. N Engl J Med 1992;327:987–92.
3. Hallstrom AP, Cobb LA, Yu BH, et al. An antiarrhythmic drug experience in 941 patients resuscitated from an initial cardiac arrest between 1970 and 1985. Am J Cardiol 1991;68:1025–31.

Chlamydia pneumoniae in Redo and First-Time Coronary Artery Bypass Graft Surgery

In their recent study, Wong et al. (1) tried to determine if *Chlamydia pneumoniae* was more prevalent in atherosclerotic blood vessels as compared with normal blood vessels of patients requiring redo and first-time coronary artery bypass graft surgery (CABG).

Using a very detailed methodologic process, they concluded that *C. pneumoniae* is not an important factor in graft failure. This very important question seems not to be definitively answered by the authors. First, the authors did not explain when the graft failed among the elective patients who had a redone CABG, knowing that a dramatic proportion of these occlusions occur soon after the operation and are attributed to technical reasons rather than atherosclerosis progression, as the authors hypothesized, even in asymptomatic patients. Second, whether or not the organism is found in plaques does not alter the basis of the infective hypothesis and atherosclerosis. That is the role of immune system activation in the presence of bacterial components (2,3). Finally, the authors considered that a few recently published, small clinical studies using antibiotics in patients with coronary artery disease were trials without a clear benefit. It is important to remember that these pilot studies are testing clinical, serologic and immunologic hypotheses, irrespective of the efficacy or safety of one particular compound (4). These results should not be used to support part of the findings of one single study.

Enrique Gurfinkel, MD, PhD, FACC
Director of Coronary Care Unit
Institute of Cardiology and Cardiovascular Surgery
Favaloro Foundation
Buenos Aires, Argentina

PII S0735-1097(99)00281-8

REFERENCES

1. Wong YK, Thomas M, Tsang V, Gallaher PJ, Ward ME. The prevalence of *Chlamydia pneumoniae* in atherosclerotic and nonatherosclerotic blood vessels of patients attending for redo and first time coronary artery bypass graft surgery. J Am Coll Cardiol 1999;33:152–6.
2. Hansson GK, Jonasson L, Seifert PS, Stemme S. Immune mechanisms in atherosclerosis. Arteriosclerosis 1989;9:567–78.
3. Gurfinkel E, Bozovich G. *Chlamydia pneumoniae*: inflammation and instability of the atherosclerotic plaque. Atherosclerosis 1998;140 Suppl 1:31–5.
4. Gurfinkel E. Open and controlled intervention trials for anusual anti chlamydia indications: asthma, atherosclerosis, myocardial infarction. In: Allegra L, Blasi F, eds. *Chlamydia Pneumoniae*. Milan, Italy: Springer-Verlag, 1999:185–7.

REPLY

Evidence that *Chlamydia pneumoniae* is associated with atherosclerosis has come from serological and pathological studies. Pathological studies have reported that *C. pneumoniae* is more common in atherosclerotic vessels as compared with normal blood vessels. This has been put forward as evidence that *C. pneumoniae* may be a cause of atherosclerosis. However, few studies have used adequate control data (1). In our study of patients needing first-time and redo coronary artery bypass graft surgery (CABG), we examined occluded venous grafts, endarterectomy specimens from native coronary arteries, new saphenous vein (SV) grafts and new left internal mammary artery (LIMA) grafts (2). Graft failure is less common with LIMA than with SV grafts, but we found that the prevalence of *C. pneumoniae* was significantly greater in new LIMA than in new SV grafts. Also *C. pneumoniae* was just as prevalent in new LIMA grafts as in failed grafts and diseased native vessels. Therefore, we concluded that *C. pneumoniae* was unlikely to be important in graft failure.

Gurfinkel implies that atherosclerosis may not have been a cause of graft failure in our study. In our unit, it is not our experience that